Chapter

Management of Odontogenic and Nonodontogenic Oral Pain

Sameer Shaikh

Abstract

Pain in the orofacial region is by far the commonest reason for patients to seek treatment. Tooth and intraoral structures are often the main sources of orofacial pain. Odontogenic pain, also commonly known as tooth pain, originates from dental structures, pulpal or periodontal. Nonodontogenic oral pain can originate from intraoral structures such as gingiva and buccal mucosa. Arriving at a correct and definitive diagnosis is of paramount importance to institute an appropriate treatment. Obtaining a detailed history from the patient including the location, duration, frequency, periodicity, character, and quality of pain assists in differentiating odontogenic from nonodontogenic causes. Wide varieties of pharmacological agents, along with invasive and noninvasive procedures, are available to manage odontogenic and nonodontogenic pain. While managing orofacial pain, clinical and pharmacological judgment should encompass a systematic and objective assessment in compliance with the strongest evidence available. In this chapter, there will be a discussion of various choices and options available to manage a few of the orofacial pain complaints.

Keywords: orofacial pain, odontogenic pain, nonodontogenic oral pain, pain management, pulpitis, periapical periodontitis, traumatic periodontitis, cracked tooth syndrome, noninfectious and nonmalignant oral ulcers, burning mouth syndrome, oral mucositis

1. Introduction

Odontogenic pain, a common malady globally and the most prevalent type of orofacial pain, originates from dental structures, pulpal or periodontal [1]. Differential diagnosis for odontogenic pain is outlined in **Table 1**. Oral pain of nonodontogenic origin can originate from the intraoral structures, such as buccal mucosa, gingival tissues, and alveolar bone. Some of the main causes for nonodontogenic pain of oral origin are shown in **Table 2**. The complexity of the orofacial region makes the management of odontogenic and nonodontogenic pain of oral origin a challenging task for the clinicians. For an effective diagnosis and treatment, the clinician should have a thorough knowledge of the various pain complaints pertaining to the orofacial region and the different options available for their optimal management [2, 3].

For managing odontogenic pain, The "3-D's" principle—diagnosis, dental treatment, and drugs—should be used. The first and foremost step is to determine the condition causing the pain and then to discover that what caused that condition. Removal of the cause usually leads to rapid recovery and should be done by an appropriate dental treatment. Medications should only be used to complement the dental treatment [4].

Origin	Possible causes
Pulpal pain	Dentine hypersensitivity Reversible pulpitis Irreversible pulpitis Cracked tooth syndrome
Periodontal pain	Periapical periodontitis • Periapical abscess • Periapical granuloma and cyst Traumatic periodontitis Periodontal (lateral) abscess Perio-endo, endo-perio, and combined lesions

Table 1.
Differential diagnosis for odontogenic pain.

• Noninfectious and nonmalignant oral ulcers
• Acute pericoronitis
• Acute alveolar osteitis (dry socket)
• Burning mouth syndrome (BMS)
• Oral mucositis (OM)
• Acute necrotizing ulcerative gingivitis (ANUG)
• Desquamative gingivitis (DG)

Table 2.
Causes of nonodontogenic pain of oral origin.

For managing nonodontogenic pain particularly in complex cases, a multidisciplinary pain management approach should be adopted encompassing both nonpharmacological and pharmacological modalities [5].

2. Odontogenic pain

2.1 Pulpal pain

2.1.1 Dentine hypersensitivity

Dissolution of the dental enamel results in development of dental caries. If caries goes unchecked, it may involve the dentin and the pulp, resulting in pain. In the initial stages, caries penetrates and exposes the dentin leading to dentine hypersensitivity. The pain due to dentin exposure is of a sharp and shooting nature with a shorter duration and is classically stimulated by exposure to heat, cold, sweet drinks/food, and mechanical trauma such as tooth brushing. Apart from caries, there exist other predisposing factors for dentine hypersensitivity. These include anatomical defects, gingival recession, erosion, abrasion, and attrition. The diagnosis of dentine hypersensitivity is based upon detection of dentin exposure or tooth wear. Therapies for managing dentinal hypersensitivity are aimed at: sealing the exposed dentinal tubules (composite resin application), reducing dentinal neuron activity (application of desensitizing agents such as potassium nitrate and strontium chloride), and making the enamel and dentin more resistant to demineralization (application of fluoride-containing medicaments) [6, 7].

2.1.2 Reversible and irreversible pulpitis

The extension of caries to pulp leads to pulpal inflammation known as pulpitis. Other cause of pulpitis can be operative dental procedures. The chemicals, heat, and friction involved in such procedures may trigger pulpal inflammation. Pulpitis has two clinical forms: acute (reversible) and chronic (irreversible). Acute pulpitis represents mild inflammation and is characteristically associated with sharp and shooting pain of a shorter duration. On the other hand, inflammation in irreversible pulpitis is severe enough to undermine the pulp. It is characterized by spontaneous and dull pain that persists even after the removal of a stimulus such as cold or heat [6–8].

Diagnosis of pulpitis is based mainly on clinical evaluation and pulp vitality tests. Radiographs can be helpful in cases where carious lesions are not clinically visible [8, 9].

The management strategies are determined based on the type of pulpitis and presence of infection involving the periapical area. In reversible pulpitis, pulp vitality can be maintained if the tooth is treated, usually by removing the caries, and then restored [10]. In irreversible pulpitis, management options include endodontic (root canal) therapy or tooth extraction. In root canal treatment, an opening is made in the tooth and the pulp is extirpated. The root canal system is thoroughly cleaned, shaped, and then obturated with gutta-percha points. Following root canal therapy, adequate healing is manifested clinically by resolution of symptoms and radiographically by bone filling in the radiolucent area at the root apex over a period of months. If symptoms persist or worsen, root canal therapy is usually repeated in case a root canal was missed [11, 12].

2.1.3 Cracked tooth or cracked cusp syndrome

Cracked tooth syndrome occurs when a crack has occurred in the enamel or dentine and reaches the pulp chamber. The crack is usually not visible to the naked eye. Application of excessive force on a normal tooth or physiologic forces applied to a weakened tooth can lead to cracks. The diagnosis of cracked tooth is often tricky. Radiography is not helpful in detection of fractures, as cracks occur in a mesiodistal direction, parallel to that of the plane of the film. Simple test is to have patient bite on a cotton roll that evokes a sharp pain. Pain due to cracked tooth is sharp and shooting in nature, and is usually associated with biting and chewing. Hot and cold stimuli also evoke the pain. Restorable teeth should be treated endodontically, followed by a full-coverage restoration of tooth. However, tooth with large cracks may require extraction [7, 13].

2.2 Periodontal pain

2.2.1 Periapical periodontitis (periapical abscess, granuloma, and cyst)

Pulpitis, if untreated, is followed by death of the pulp. The necrotic pulp is infected and leads to spread of infection through the apical foramina into the periapical tissues. This in turn causes inflammation and destruction of the periradicular tissues known as periapical periodontitis. It includes acute/chronic nonsuppurative inflammation and suppurative inflammation. Periapical granuloma forms due to chronic inflammation without pus, while periapical abscess is the result of inflammation involving pus. The other likely cause of periapical periodontitis can be chemical irritation. This irritation can be due to the escape of antiseptics used for root canal sterilization through the root apex into the surrounding periapical area [11, 12].

Acute periapical abscesses characteristically present with severe pain in the area of the nonvital tooth particularly on percussion, inflammation, or complaint of pus drainage (with its associated foul taste). Pain also typically interferes with sleep. Treatment includes drainage through an opening in the tooth itself or through the soft tissue surrounding the jaw, if cellulitis has developed. If patients with abscess have systemic signs of infection (e.g., fever), an oral antimicrobial is prescribed (amoxicillin 500 mg every 8 hours; for patients allergic to penicillin, clindamycin 150 or 300 mg every 6 hours). On resolution of the abscess, the patient should undergo root canal therapy or extraction [8, 10, 11, 14].

Periapical granulomas or cysts usually follow acute pulpal infection that remains unresolved due to inadequate drainage. Tooth with periapical granulomas may present with a dull pain or may be asymptomatic. Radiographically, abscesses, granulomas, or cysts have the same features and microscopic examination should be done for distinction. Teeth with periapical granulomas are nonvital and needs root canal treatment or removal. Root canal treatment done competently leads to healing even if cystic phase has started. Persistence of periapical radiolucency after 6–12 months may be due to technical faults associated with root canal treatment. In such a case, apical curettage with apicoectomy may be indicated [6, 8, 14, 15].

2.2.2 Traumatic periodontitis

Traumatic periodontitis is a painful condition that arises because of injury to the periodontium. This injury is caused by the trauma from excessive occlusal forces. The occlusal trauma affecting periodontium can be primary, secondary, or combined. Tooth or teeth with normal periodontal support enduring increased occlusal loads may undergo primary occlusal trauma. The causes may include bruxism, overextended margins of restorations, excessive loading during orthodontic movements, and recent fitting of a new partial denture. Tooth or teeth with inadequate periodontal support if subjected to normal occlusal forces may undergo secondary occlusal trauma. Excessive occlusal force on a diseased periodontium may lead to combined occlusal trauma. The excessive occlusal forces are generally from parafunctional movements such as bruxism. The clinical features of traumatic periodontitis include pain on chewing/biting or percussion, progressive tooth mobility, and nonphysiological movement of tooth during function (fremitus). Additionally, there can be gingival inflammation with pocket formation in combined occlusal trauma.

Radiographic features include evidence of circumferential and furcal bone loss, in combination with widening of the periodontal ligament space.

The goal of management of traumatic periodontitis is the removal of excessive occlusal forces and brings the dentition in occlusal harmony. Primary occlusal trauma can be managed with analysis and correction of occlusion. One or more of the following steps can do occlusal adjustments: tooth movements, tooth removal, dental restorations, coronoplasty, etc. Progressive tooth mobility due to secondary occlusal trauma may be reduced by occlusal adjustment. Pain occurring due to hypermobility can be managed by splinting of teeth. The aim of splinting is to increase the resistance of dentition to the occlusal forces through stabilization. It involves joining of two or more teeth [16, 17]. Managing the periodontal inflammation is of primary importance in cases of combined occlusal trauma. Premature occlusal contacts usually contribute to the progression of periodontitis. This can be tackled by simple correction of the occlusion that may eradicate the premature occlusal contacts [16, 17].

2.2.3 Periodontal (lateral) abscess

A periodontal abscess arises because of acute infection of a periodontal pocket. Unlike a periapical abscess, periodontal abscess is associated with a vital tooth.

Varieties of reasons are implicated in causation of periodontal abscess. Primarily incomplete calculus removal can be a causative factor. Occasionally, it may occur following root planing, as the trauma to pocket lining implants bacteria into the periodontal tissues. Other contributing factors can be food packing down between teeth with poor contact points or foreign body (e.g., fish bone) driven through the floor of a pocket. Poorly controlled diabetes mellitus can also be a predisposing factor for periodontal abscess formation.

Periodontal abscess has a rapid onset. The gingival swelling and inflammatory edema prevent drainage through the pocket orifice. The initial gingival tenderness progresses to throbbing pain that is well localized. The affected tooth is tender to percussion or biting. There is tooth mobility with its elevation in the socket. Pus exudation may occur from the pocket; however, a deep abscess has a sinus tract that points on the alveolar mucosa. Fever and regional lymphadenopathy can be occasional clinical features. The vitality of the tooth, deep pocketing, and less severe tenderness helps to differentiate between a periodontal and pulpal abscess.

Periodontal abscess should be ideally drained through pocket or occasionally by an incision through the gingiva. If the abscess is too large and drainage cannot be done, subgingival scaling and root planing or deferring the surgical access until the major clinical signs have subsided. Before initiating the treatment of acute periodontal abscess, the evaluation of patient's medical history, dental history, and systemic conditions is crucial to determine the need for antibiotics. The indications for antimicrobial therapy in patients with acute abscess are fever, lymphadenopathy, evidence of spreading of infection (cellulitis), deep periodontal pocketing, and immunosuppression. Administration of antibiotics alone without the local drainage of the abscess is contraindicated. The drainage is mandatory in order to eliminate the etiologic factors. Extraction of the affected tooth can be considered as a last resort to treat the periodontal abscess, if there is poor response to therapy, horizontal tooth mobility exceeding 1 mm, pocketing exceeding 8 mm, and more than 40% alveolar bone resorption [6, 18, 19].

2.2.4 Perio-endo, endo-perio, and combined lesions

In perio-endo lesions, microorganisms from the periodontal pockets can reach the pulp through accessory canals, thereby leading to pulpal inflammation and necrosis.

In endo-perio lesions, pulpal necrosis leads to involvement and destruction of the periodontal ligament and adjacent alveolar bone. Clinically endo-perio lesions present as deep periodontal probing depth extending to the apex of the tooth.

In managing the lesions of pulpal or periodontal origin, making an accurate diagnosis as to the source of infection is a critical determinant of the treatment outcome. Sequence of the disease process can be an important factor in determining the exact nature of lesions: perio-endo and endo-perio lesions. Conventional root canal therapy (RCT) alone leads to a complete resolution of the periodontal defects arising from primary pulpal infection. However, pulpal infections resulting from primary periodontal infections require both endodontic and periodontal treatments for achieving complete healing [18].

3. Nonodontogenic pain of oral origin

Oral ulcers are a broad entity that encompasses a variety of causes, such as infections (bacterial, viral, and fungal), neoplasia, immunological disturbances, drug reactions, etc.

3.1 Noninfectious and nonmalignant oral ulcers

A detailed clinical history and examination, and laboratory investigations including biopsy, culture, and immunochemistry tests are essential for ruling out the neoplastic, infectious, and immunological causes of oral ulcerations. The causative factors for noninfectious and nonmalignant oral ulcers usually include mechanical trauma (self-induced trauma such as on chewing and biting, aggressive tooth brushing, and iatrogenic causes particularly due to dental treatment) and chemicals (aspirin, acetylsalicylic acid, acid etchants, etc.)

Superficial ulcers usually lead to soreness; severe pain and discomfort are the features of deep ulcers.

On elimination of cause, acute forms of traumatic- and chemical-induced ulcers usually heal in 7–10 days. They develop chronicity if subjected to continuous trauma or irritation. The considerations in management of such type of ulcers are as follows:

1. Maintenance of oral hygiene. In the presence of an ulcer, tooth brushing particularly near to the ulcerative area can be detrimental. In such as case, an antiseptic mouthwash (e.g., 0.2% chlorhexidine solution) can be of considerable help. Chlorhexidine mouth rinse is recommended to be used three times daily after meals and held in the mouth for at least 1 minute. Oral rinsing with chlorhexidine has been found to lessen down the discomfort and duration of aphthous stomatitis.

2. Avoidance of irritation or injury to the area of ulceration. Covering agents, e.g., carboxymethylcellulose paste (Orabase®) and carmellose sodium can be helpful in safeguarding the ulcers from the effects of friction or injury. When correctly applied, these covering agents absorb moisture and form an adhesive gel, which can remain in place for several hours.

3. For management of pain, over-the-counter anesthetic agent (an example is Orobase® with 20% benzocaine). Topical application of weak potency corticosteroids (hydrocortisone hemisuccinate) and medium potency steroids (triamcinolone acetonide) also assist in reducing the associated pain and inflammation; however, they are unlikely to expedite the healing of ulcers. Hydrocortisone hemisuccinate 2.5 mg pellets allowed to be dissolved in the mouth close to ulcers, three times a day. Triamcinolone 0.1% in Orabase applied to ulcer three times daily. However, long-term and/or repeated topical application of such corticosteroids has a downside in the form of adrenal suppression. This concern can be addressed by using the topical corticosteroids at the lowest possible concentration and frequency. The problem of adrenal suppression is not evidenced with 0.05% fluocinonide in adhesive paste and betamethasone-17-valerate mouth rinse.

Tetracycline (e.g., doxycycline), or tetracycline plus nicotinamide in rinse form may provide significant pain relief and reduce ulcer duration, particularly in aphthous ulcers. However, usage of tetracycline should be avoided in children below 12 years of age due to the risk of tooth staining. For oral rinsing, a tetracycline capsule (250 mg) is crushed and stirred in a little water and held in the mouth for 2–3 minutes, three times daily.

Tetracycline mouth rinses can also reduce the frequency of aphthous ulcers on regular usage for 3 days each week.

Salicylates based on their anti-inflammatory role can be helpful in reducing the discomfort of oral ulcers. Over the counter, preparation of choline salicylate in gel form is recommended for application to ulcers, 3–5 times daily [6, 20, 21].

3.2 Acute pericoronitis

It is the inflammation of the flap of tissues (operculum) around an erupting tooth, and most commonly associated with impacted mandibular third molars. The chief complaints in this condition are severe pain that can radiate to surrounding areas and swelling of the pericoronal tissues. The hyperplastic-inflamed flap of tissue can become a hotbed for bacteria, as it readily holds food particles and debris. This scenario leads to bacterial infection with clinical manifestations of discharge of pus, trismus, fever, regional lymphadenopathy, and in some cases spread of the infection to adjacent tissue spaces.

If the pain and inflammation are limited to the tooth, local measures, such as debridement of food debris and plaque, irrigation with normal saline or hydrogen peroxide, and avoidance of occlusal trauma are recommended.

Antimicrobial therapy is indicated for patients presenting with fever, trismus, and pus exudation. Metronidazole 400 mg three times a day for 5 days is to be prescribed in combination with phenoxymethylpenicillin 500 mg four times a day for 5 days.

If it is envisaged that the tooth can be useful for chewing and patient also has the desire to retain the tooth, hyperplastic pericoronal tissue should be excised out through a minor oral surgery procedure known as operculectomy. This will allow better access to properly clean the area and prevent the accumulation of bacteria and food debris. In some unfortunate instances, the gum tissue may grow back and create the same problem.

Since impacted teeth frequently are unfavorably aligned and do not erupt completely, extraction of such tooth is commonly performed. This method eliminates any chance of recurrence of pericoronitis.

The risks and benefits of removal of impacted molars are mired in controversy, as extraction can lead to inferior alveolar nerve damage; retention can precipitate serious, even life-threatening infection [14, 22].

3.3 Acute alveolar osteitis (dry socket)

This painful condition is a complication that may occur following dental extraction. It presents with a severe throbbing pain caused by bone exposure at the site of extraction. Following the extraction, a blood clot forms within the extraction socket to safeguard the bone. If a blood clot forms inadequately in the socket or it is dislodged, the bone and nerves are exposed, leading to pain. Smoking, excessive extraction trauma, difficult disimpactions of third molars, vasoconstrictor in local anesthetic, and oral contraceptives are some of the predisposing factors to alveolar osteitis. Alveolar osteitis can strike 3–5 days after an extraction and may persist for a week. The exposed bone is acutely tender to touch; hence, mechanical stimulation by tongue movement and food particles results in frequent acute pain. On clinical examination, the socket appears empty with visible bony lamina dura.

Minimization of trauma related to the extraction procedure can be an important factor in prevention of dry socket. Since removal of the debris from the socket expedites healing, irrigation with warmed saline or chlorhexidine is suggestive. Use of intra-alveolar dressing materials such as bismuth iodoform paraffin paste and lidocaine gel on ribbon gauze can protect the socket from painful stimuli and collection of food debris. These dressing materials also impart a soothing sensation of warmth in the painful area. Usually after one or two dressings, significant pain relief is achieved. It is better to be on the lookout for signs of infection, such as pus in the socket, localized swelling, and lymphadenopathy. Antibiotics should be prescribed if these signs are there. It is crucial that

the reason for infection is determined such as retained root or bony fragments. A radiograph can be helpful. Surgical extraction is indicated for removal of root tip or bone sequestrum [6, 13, 23].

3.4 Burning mouth syndrome (BMS)

Burning mouth syndrome (BMS) is a complex painful disorder that is characterized by warm, burning, or tingling sensation in the oral mucosa, tongue, or lips. The pain may be associated with a feeling of intermittent numbness. Other associated features may include metallic taste and dryness in the mouth. Interestingly, a variety of names has been associated with this condition such as oral dysesthesia, stomatodynia, glossodynia, stomatopyrosis, glossopyrosis, sore mouth, and sore tongue. BMS is a reasonably common chronic complaint to affect middle age or elderly patients, especially females. Diagnosis of BMS is challenging, because usually no clear-cut dental or medical cause is evident and laboratory findings does not reveal any abnormality.

BMS can be classified into two clinical variants, namely, primary and secondary BMS. If no underlying medical or dental problem becomes evident on investigations, the diagnosis is primary or idiopathic BMS. Probably, the damage to the nerves that control pain and taste leads to primary BMS. Secondary BMS is caused by local, systemic, or psychological factors. A few common causes of secondary BMS include, dry mouth, acid reflux, deficiency of iron or vitamin B, hormonal disturbances (such as from thyroid problem or diabetes), etc. Because burning mouth syndrome can be associated with a wide array of local, systemic, or psychological conditions, an ambitious diagnostic approach is warranted. This approach should be based on a detailed history, clinical examination, laboratory tests, and exclusion of all other possible oral and systemic problems.

If no organic cause can be found and diagnosis suggests psychological factors such as anxiety, stress, and depression, it is advisable to make the patient aware by explaining that depression and other emotional disturbances are just as much illnesses and cause as much suffering as physical diseases. Apart from psychogenic medications, cognitive behavioral therapy is indicated in BMS.

Depending on the causative factors, medications used for BMS include antidepressants, analgesics, antiepileptic, antifungal, antibacterial, sialagogues, antihistamines, anxiolytics, antipsychotics, and vitamin, mineral, and hormonal replacements.

The topical application of clonazepam (by sucking a tablet of 1 mg), three times a day for 14 days can reduce the burning symptoms. *Aloe vera* gel also helps to reduce the burning sensation and pain in the sore areas of the tongue. Symptoms of secondary BMS go away when the underlying medical condition, such as diabetes or acid reflux, is treated [24, 25].

Overall, successful management of BMS is dependent on a holistic diagnostic workup and collaborative management involving dental practitioners, psychologist, and physician.

3.5 Specific anticancer treatment painful oral complications: oral mucositis (OM)

This grossly painful disorder usually occurs as a complication of chemo- and radiotherapy. An allergic reaction to certain medications, dental materials, or infections may also lead to nonspecific mucositis. Oral mucosal injury is the hallmark of OM that occurs due to the interference of chemotherapy and/or radiation therapy with normal turnover of oral mucosal cells.

Chemotherapy-induced and/or radiation therapy-induced OM clinically manifests as the painful swelling, atrophy, and ulceration of the oral mucosa. *Candida*

and streptococcal infections may also occur due to the disintegration of the oral mucosa.OM-affecting pharynx and other areas of alimentary canal can lead to complications, including dysphagia, electrolyte disturbances, systemic infection, malnutrition, and even death [20, 26].

Oral mucosal injury tends to be acute in cases where chemotherapy is administered over a short span of time. Chemotherapy-induced mucosal damage usually develops within a week after the start of therapy and peaks within 2 weeks. Radiotherapy-induced mucositis has a slower onset since it is most often administered in small fractions given over weeks. Radiation-induced mucositis typically starts in 1–2 weeks of starting the radiotherapy at cumulative doses of about 15 Gy (gray, a unit of absorbed radiation). At doses greater than 30 Gy, OM attains full severity and may last for weeks or even months.

Factors related to treatment and patient characteristic can influence the development of OM. Treatment factors that influence the severity and presence of mucositis include the class, dose, and administration frequency of systemic chemotherapeutic agents, radiation dose and field, and use of adjuvant chemotherapy and radiation. The majority of patients treated for head and neck cancers or those receiving high-dose chemotherapy develop severe OM. Usually the healing within lesions of OM is evidenced within 2–4 weeks after stoppage of either therapy.

So much so, the OM is a painful and agonizing condition that it has a drastic impact on oral hygiene maintenance, nutritional intake, and quality of life. Current clinical management of OM is largely supportive and aimed at maintaining oral hygiene, pain relief, and nutritional support. A majority of patients with mucositis pain has difficulty in food intake through mouth and a nasogastric tube or gastrostomy tube helps to achieve nutrition. Diet modifications in the form of liquid and soft diet are suggested to facilitate the food intake during the cancer therapy [27].

Maintenance of oral hygiene has an important contribution in the prevention and management of OM; however, it remains a neglected habit. Moreover, a good oral care helps to prevent secondary infection and sepsis in the lesions of OM. Oral hygiene measure such as tooth brushing, flossing, rinsing with sterile water, and using mouth moisturizers helps control pain and bleeding and prevent infections of the oral soft tissue. However, at the same time, caution must be exercised that tooth brushing and flossing do not traumatize the oral mucosa. In case, a patient with OM is unable to tolerate the use of a tooth brush, oral sponges and foam brushes can be used instead.

Simple analgesia, e.g., paracetamol (1 g four times a day) in soluble form used as a mouth rinse will be adequate to control the mild-to-moderate pain of OM. For controlling severe pain, opioid analgesics (e.g., hydromorphone or morphine) can be used. Use of opioids is both logical and appropriate to alleviate the intolerable pain of OM, and strong opioids can be helpful in this direction.

When swallowing pills can be problematic in patients with severe OM, the use of parenteral administration of opioid analgesics is required. For seeking short-term relief in pain of OM, oral rinsing with 2% viscous lidocaine (topical anesthetic) in combination with diphenhydramine and magnesium aluminum hydroxide may allow the patient to eat and maintain oral hygiene. Mucosal-coating agents such as sucralfate, Gelclair®, and Caphosol® by adhering to oral mucosa form a protective coating. This coating aids in patient comfort by shielding the exposed and overstimulated nerve endings [20].

Prevention of OM is also an important aspect to be considered and has involved multiple medications. The updated clinical practice guidelines for the prevention and treatment of mucositis have suggested the use of chemo-preventative agents to prevent and/or reduce severity of OM. The most commonly prescribed preventative agents for OM are ice chips (given 30 minutes prior to chemotherapy)

or amifostine (a thiol drug) and keratinocyte growth factor-1 (palifermin). Moreover, the Multinational Association of Supportive Care in Cancer (MASCC) and the International Society for Oral Oncology (ISOO) guidelines for treatment of oral recommends the use of benzydamine for prevention of radiation-induced OM. Benzydamine hydrochloride (HCl) is a cytoprotectant with analgesic, anti-inflammatory, and antimicrobial activity. On being used as an oral rinse, it significantly reduced OM-related erythema and ulceration [20].

3.6 Acute necrotizing ulcerative gingivitis (ANUG)

Acute necrotizing ulcerative gingivitis (ANUG) is an acute infection of the gingiva and is characterized by pain, bleeding, fetid breath, and gingival necrosis. Fever, malaise, and regional lymphadenopathy may be accompanying features. Oral functions including speaking and swallowing become difficult due to intense gingival pain.

Destructive pattern in the form of gingival ulcerations, necrosis, and 'punched-out' ulcerated papillae makes ANUG unique when compared with other periodontal diseases. Initially cratered ulcers affect the tips of interdental papilla, later on spreading along gingival margins. ANUG most commonly affects smokers and stressful immunocompromised individuals. Other risk factors are neglected oral hygiene, sleep deprivation, and malnutrition. ANUG is an opportunistic bacterial infection that is caused by a complex of *fusiforms* and *spirochaetes*.

Maintenance of oral hygiene through self-care and gentle debridement by the dentist is the stepping stone to the successful management of ANUG. Patients may be advised to use mouth rinses, such as warm normal saline or 1.5% hydrogen peroxide or 0.12% chlorhexidine at hourly intervals for the first few days. Analgesics may help to ward off the intense pain associated with ANUG. In order to prevent recurrence of ANUG, the patient must be educated to maintain high personal oral hygiene, to have adequate nutrition, and to get sufficient rest. Antibiotics are indicated in case of systemic involvement. The recommended antibiotics are amoxicillin 500 mg, three times daily for 10 days plus metronidazole 250 mg, three times daily for 10 days. The healed gingival craters can act as stagnation areas where plaque can accumulate and ANUG may reoccur. For correction of superficial craters, gingivectomy and/or gingivoplasty procedures may be helpful. For rehabilitation of deep craters, periodontal flap surgery or regenerative surgery may be considered [28–30].

3.7 Desquamative gingivitis (DG)

Desquamative gingivitis (DG) is a specific clinical presentation of unknown etiology in which the attached gingiva appears fiery red, glazed, and friable. Desquamative gingivitis may be a clinical manifestation of various mucocutaneous disorders—erythema multiforme, erosive lichen planus, pemphigus, pemphigoid, and psoriasis. DG is characterized by gingival soreness and burning sensation, which worsens on eating spicy and acidic food. The typical clinical feature in severe cases is of desquamation of gingival epithelium. The treatment of DG is aimed at minimizing the gingival injury and irritation. Therefore, the patient should avoid spicy or acidic foods. Oral hygiene maintenance can be helpful in removal of exacerbating factors, particularly dental plaque. However, in order to avoid injury to the friable gingiva, tooth brushing should be done gently with a soft tooth brush or toothette. Use of an anesthetic mouthwash, e.g., benzydamine hydrochloride can be helpful in tackling the pain. Topical therapies are the mainstay of treatment for DG. High potency corticosteroid gels are commonly used as first-line topical therapy. Clobetasole-17-propionate or fluocinonide 0.05% in gel form can be prescribed. Ease of gel application can be facilitated via the use of custom fabricated

trays. Furthermore, 0.1% triamcinolone orabase can also be used. For complete resolution of DG, it is important that the underlying disease leading to DG is diagnosed and treated appropriately by specific therapies [31, 32].

4. Conclusion

Odontogenic and nonodontogenic pain may occur due to a variety of factors and causes. A differential diagnosis of orofacial pain, distinguishing between odontogenic pathologies and nonodontogenic painful etiologies, is a requisite before taking any clinical or pharmacological decision for pain management. Exactness of differential diagnosis is dependent on a thorough medical and dental history, comprehensive clinical examination, and appropriate investigations. Any decision on pain management should encompass a treatment regimen (e.g. palliative, dental, pharmacological, and psychological) that can adequately address the clinical problem of pain. For the successful accomplishment of a durable pain management, the treatment decisions should be based upon the best-available evidence, consideration of cost-effectiveness, and patient's expectation. Specialist referral is warranted, if the conventional clinical and pharmacological measures fail to control the odontogenic or non-odontogenic oral pain [1].

Conflict of interest

The author declares no potential conflicts of interest with respect to the authorship and/or publication of this chapter.

Author details

Sameer Shaikh
Department of Oral Diagnosis and Oral Medicine, University of Ha'il, Ha'il, Saudi Arabia

*Address all correspondence to: smrshaikh@gmail.com

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